REMARKS

Claims 4-7 and 10 remain active in the case. Reconsideration is respectfully requested.

Applicants' representative wishes to thank Examiners Bunner and Kemmerer for the courteous and helpful discussion of October 7, 2004. As a result of the discussion, it is believed that the issues in the case have been clarified and that the prosecution of the application has been materially advanced.

Claim Amendment

Claim 10 has been amended in order to specify a method of treating a subject suffering from multiple sclerosis rather than a method of preventing multiple sclerosis. Accordingly, the only change that has been made to the claim is to redefine the scope of the claimed method. Entry of the amendment is respectfully requested.

Invention

The present invention is directed to a peptide sequence, a pharmaceutical composition containing the peptide and a method of treating a subject suffering from multiple sclerosis. The peptide sequence is as follows: of the present invention are selected from the group consisting of:

R-Asn-Gly-Val-Gly- His- Gly-Phe-Gly-Asn-Gly-Val-Gly-Pro-Gly-Thr-Gly-Pro-Gly-Ser-Gly-R (I) (SEQ ID NO: 1)

Claim Rejection, 35 USC 112, First Paragraph

The Examiner, in raising the issue of a lack of satisfactory enablement on the part of the present specification for the invention as claimed, reverts to statements in the Hart et al publication to the effect that animal models which exhibit CNS inflammation and demyelination do not fully account for the irreversible neurological deficit in advanced MS and that the neurological deficit in MS and EAE is still poorly understood. The Examiner then states that a large quantity of experimentation would be required apparently to lead the

skilled artisan to a conclusion that a particular therapeutic regimen would be of benefit in treating humans who suffer from MS; and further refers to the statement at the bottom of column 1, page 379 of the desire for evidence from preclinical models that are more closely related to the human disease than is provided by the test results obtained from rodent models.

While it is true that there are difficulties in extrapolating the results obtained from rodent models to an effective treatment of the disease of MS in human beings, nevertheless, as the reference states in the first paragraph of column 1 of page 377:

> -- In general these mouse strains (transgenic mice of the C57BL/6 background) provide powerful new approaches to study detailed pathological mechanisms, factors, and pathways that could provide specific targets for therapeutic strategies. --

Further, the publication states in column 1 of page 379 that:

> -- In conclusion, rodent models of MS have been critical for the study of the basic mechanisms of recruitment of inflammation within the CNS as well as mechanisms leading to myelin and axonal damage.--

Clearly, at the very least, rodent models are of very important benefit in providing an indicator of the possible effectiveness of a given proposed therapeutic regimen. In the present specification, Example 1 describes an assay test involving the peptide of formula I that is presently claimed in which the peptide was tested in four groups of SJL female mice at the age of 6-15 weeks. (This strain of mice was genetically selected for its ability to develop EAE.) The results obtained as presented in Table 1 of the text show that for seventeen mice who had been immunized by the intraperitoneal administration of the claimed peptide of the invention of formula I and then administered P81-100 to induce EAE, none exhibited EAE. While this model and results obtained may not be a completely reliable indicator of the effect the peptide may have when administered to human beings suffering from MS, nevertheless, the model employed and result obtained are sufficiently suggestive of a possible therapeutic use in the treatment of human beings. Applicants, in fact, note the definitive statement in the MPEP of Section 2107.03 (I) that:

> -- As a general matter, evidence of pharmacological or other biological activity of a compound will be relevant to an asserted therapeutic use if there is a reasonable correlation between the activity in question and the asserted utility. An applicant can establish this reasonable correlation by relying on statistically relevant data documenting the activity of a compound or composition, arguments or reasoning, documentary evidence or any combination thereof. The applicant does not have to prove that a correlation exists between a particular activity and an asserted therapeutic use of a compound as a matter of statistical certainty, nor does he or she have to provide actual evidence of success in treating humans where such a utility is asserted. Instead, as the courts have repeatedly held, all that is required is a reasonable correlation between the activity and the asserted use. --

The MPEP in Section 2107.03 (III) then goes on to say:

> -- If reasonably correlated to the therapeutic or pharmacological utility, data generated using *in vitro* assays, or from testing in an animal model or a combination thereof almost invariably will be sufficient to establish therapeutic or pharmacological utility for a compound, composition or process. –

Applicants submit that, in fact, they have provided in the text of the specification a reasonable correlation between the stated therapeutic utility of a method of treating a subject suffering from MS and the data of the assay conducted with the peptide of formula I. Accordingly, the rejection that has been raised under 35 USC 112 is believed obviated and withdrawal of the rejection is respectfully requested.

It is believed that the application is in proper condition for allowance. Early notice to this effect is earnestly solicited.

Respectfully submitted,

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